tened. Ischemic softening of the posterior knee of the capsula interna and of the ventral portion of the corpora quadrigemina were present. The brain and cord, studied in serial sections microscopically, gave in brief the following results: The softening observed macroscopically was microscopically demonstrated, as also the inferior lip of the calcarine fissure at the gyrus lingualis. As a result of the softening of the Ammonshorn and the fascia dentata the fornix in its greatest extent showed descending degeneration of its fibers. Secondary degeneration was found in the pyramidal tract and in the foot of the eruri cerebri the tract joining the parietal lobe with the cerebellum. The latter degeneration could be followed through the pons to the hemisphere of the cerebellum of the other side. The medial bundle of the foot of the crura remained unaffected. The degeneration was especially well marked in the parietal lobe. A portion of the lateral ventricle wall, the projection strata of the parietal lobe to very nearly the level of the incoming fibers of the commissura anterior were degenerated. The degeneration of the projection system in the parietal lobe was in continuity with the degenerated portion of the corpora quadrigemina. In the medulla the middle and outer portion of the "Striekkorper" and the external and internal arcuate fibers, the cerebellar olivary tract, including the external region of the right and the internal hilus of the left olive were degenerated. In the cervical cord the pyramids, the territory of the periphery were clearly degenerated. As a result of the cerebellar defect, a descending degeneration of the lateral peripheral fibers of the same side of the cord and to a less extent on the other side, were found degenerated. On the same side the Clarke cells were atrophied and in part absorbed. The relation of the clinical symptoms to the anatomical condition is stated as follows: The left-sided paresis is an evident result of the disturbance to the internal capsule and the degeneration of the pyramids, in the same way the loss of cutaneous and deep sensibility is due to the break in conduction in the fibers of the posterior knee of the internal capsule. The ataxia and the tremor, suddenly appearing on the right side, before this intact, are due in all probability to a fresh injury of the cerebellum, the attack being ushered in by vomiting and vertigo. SCHWAB.

NATURE ET TRAITEMENT DE LA MYELITE AIGUE (Nature and Treatment of Acute Myelitis). G. Marineseo (Nouvelle Iconographie de la Salpêtrière, 13th Year, No. 6, Nov.-Dec., 1900, p. 561).

Every acute myelitis is characterized by multiple processes of reaction from the side of the vessels, interstitial tissue and nerve cells. The two first constitute the active processes of multiplication and proliferation, giving rise in this way to more or less extensive foci and to nodules.

The phenomena of reaction on the part of the nerve cells lead rapidly to their degeneration. The apparent lesion which is commonly seen in every acute myelitis is hemorrhagic softening, a lesion which is the consequence of circulatory disturbances caused by toxic and infectious agents, the only factors which cause an acute myelitis.

Following the extent and the topography of these vascular lesions, it can be admitted with Goldscheider and Leyden that there are several varieties:

Transverse myelitis, diffuse ascending and descending myelitis, disseminated myelities, and poliomyelitis.

The vascular and infectious nature of this last has been brought

to light by Pierre Marie, whose ideas on this subject Marinesco fully accepts.

The infectious and toxic nature of acute myelitis admitted by Pierre Marie has actually been demonstrated by pathologic examination and by bacteriologic and experimental researches. I have had occasion to examine six cases of acute myelitis, and in two of these I have determined the presence of streptococci; in a third the pneumococcus; while a fourth showed a microbe resembling the bacillus of malignant edema. In the fifth neither lumbar puncture nor histologic sections showed the presence of any organism.

This latter is no proof against the infectious nature of myelitis, because experimental researches have shown that the organisms disappear from the cord at the end of a few days. Thus in my fifth case, in which the myelitis had begun acutely, the patient did not die until three months after its first appearance. In the sixth case it was a question of a myelitis, which I have described for the first time, in a disease of young dogs.

The myelitis, or rather the meningo-myelitis, which I have been able to diagnose in this case, presents itself more in the form of separated foci, following more particularly the course of the anterior and posterior root arteries. In a number of cases of infantile paralysis which I have had occasion to study, the myelitic foci were situated always in the course of the ramifications of the artery of the anterior fissure. Sometimes all these ramifications are attacked and the foci of poliomyelitis involves the anterior horn in its totality. At other times it is only the artery which nourishes the antero-external group, the middle group, and very rarely the antero-internal group, which is attacked.

I have never found organisms in the foci of infantile polio-myelitis which could easily be foreseen.

Landry's ascending paralysis is most often dependent upon a diffuse ascending and descending infectious myelitis. My own studies, together with those of Pierre Marie, Oettinger, the case of Ballet and Dutil, have shown this. Sometimes it depends upon a polyneuritis (Dejerine, Kahler, Pitres, Vaillard and Raymond). I have myself seen a very remarkable case of this kind.

The majority of microbes are capable of producing myelitis. The first to be cited are the streptococci, the agent of rabies, the pneumococcus, and other organisms. I have been able to produce myelitis experimentally in four different ways:

(1) By the injection of an organism in a blood vessel distant from the cord. (2) By a blood vessel supplying it directly, following the procedure of Lamy. (3) By the nervous path (innoculation in the sciatic nerve); and lastly by the introduction of microbes in the spinal canal. The results are variable, depending upon the operative method which is followed. The injecting of organisms by a vessel distant from the cord produces rarely a myelitis, and when it does it is not very marked. The same injection into the arteries of the cord causes a polio-myelitis. The innoculation in the sciatic nerve causes a meningo-myelitis, more marked on the side of the nerve injected. While the injection into the spinal canal occasions a bilateral meningo-myclitis, very considerable at the level of the injection and diminishing in the ascendent and descendent sense. According to the vascular system affected, there can be observed from these experiments a myelitis, a disseminated myelitis, the polio-myelitis in foci, analogous to that of infantile paralysis.

The appearance of the phenomena characterizing myelitis can be

found by freezing the vertebral column, by local traumatism, etc. There are in all myelitis two leucocytic reactions: (1) A precocious reaction of defence, which consists in the migration of mononuclear and polynuclear cells, a reaction whose purpose is to vanquish the organism. (2) A tardy leucocytic reaction, the purpose of which is to carry away the products of degeneration resulting from the myelitic process.

As a specific treatment I have employed the serum of Mamorek in myelitis due to the streptococci, but without definite results. I have found the same lack of success in the employment of methylene blue in two cases of meningo-myelitis. Against the violent pains which one of my patients experienced I have used intradural injections of cocain with a certain degree of success. SCHWAB.

PSYCHIATRY.

FRIEDRICH NIETZSCHE: A STUDY IN MENTAL PATHOLOGY. William W. Ireland (The Journal of Mental Science, Vol. 47, 1901, p. 1).

The author has here given another interesting pen picture of mental malady in a man of mark. They are always well done and the delineation of Nietzsche's gradual mental disintegration is fascinatingly portrayed. It would be impossible to summarize the entire paper, it should be read through, but, Dr. Ireland says, in one place, that the unfortunate man was born with a hereditary tendency to an abnormal mental action; in infancy he was backward; in childhood he was shy and solitary; in youth he took no pleasure in the sports and amusements of young men, but he was quick at book-learning and literary aptitude with a love for straying away from beaten paths. A careful education by a good mother helped to keep down his lower propensities, and the early dignity of a responsible position and academic surroundings made him give hostages to good behavior. But he soon showed an irrepressible combativeness and an excessive self-conceit. The connection of his nervous sufferings with his mental derangement is not clear, but no doubt these exasperated him and increased his discontent with life. His was the condition described as *folie de doute*, anguish of doubts. The restless working of his intellect was always accompanied by exaltation of the affective faculties, the power of correct reasoning slowly decayed and the bonds of restraint became weaker. His aggressiveness and egotism became more and more prominent. "The peculiarity of his insanity seems to have been that while he retained sufficient powers of self-restraint to refrain from breaking through outward rules of conduct within his limited sphere of intercourse with older men, he gratified his extravagant propensities by writing reckless and provocative books against the beliefs which were most cherished by those amongst whom he dwelt." JELLIFFE.

BEITRAG ZUR KENTNISS DER TYPHUSPSYCHOSEN (Contribution to the Knowledge of Psychoses in Typhoid Fever). Deiters (Münchener med. Wochenschrift, 1900, xlvii. 47, p. 1623).

The author gives the history of two cases, which he thinks belong to the form of "initial delirium" in typhoid described by Kraepelin and by Aschaffenberg. They occurred in a brother and sister from a locality in which typhoid was prevalent, and having a strong hereditary predisposition to insanity.

Case I.—The brother, twenty-four years old, was taken sick with fever and vomiting. These symptoms subsided after two days, but